BIOCHEMICAL AND GENETIC ASPECTS IN THE PATHOGENESIS OF CARDIOVASCULAR EVENTS AND NON-ALCOHOLIC FATTY LIVER IN YOUNG ADULTS WITH SPECIAL EMPHASIS ON ADIPONECTIN ENCODING (ADIPOQ) GENE EXPRESSION

A SYNOPSIS

Submitted by

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BONAFIDE CERTIFICATE

This is to certify that this synopsis of the thesis titled "**BIOCHEMICAL AND GENETIC ASPECTS IN THE PATHOGENESIS OF CARDIOVASCULAR EVENTS AND NON-ALCOHOLIC FATTY LIVER IN YOUNG ADULTS WITH SPECIAL EMPHASIS ON ADIPONECTIN ENCODING (ADIPOQ) GENE EXPRESSION**" is the bonafide work of **RACHANA RAVEENDRAN (Registration No.: 21901008)** towards the fulfillment of the requirements for the award of the Doctor of Philosophy.

It is certified further that the work reported herein is the genuine and original work based on the results of the experiments and investigations carried out independently by the scholar during the period of study under my guidance and supervision.

This work submitted for consideration is original and does not form part of any other thesis or dissertation on the basis of which a degree or award was conferred on an earlier occasion.

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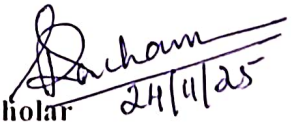

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ABSTRACT

Cardiovascular disease (CVD) and metabolic dysfunction-associated steatotic liver disease (MASLD) are increasingly prevalent among young adults due to obesity, insulin resistance, and inflammation. Oxidative stress and cytokine imbalance exacerbate vascular and liver damage. The ADIPOQ gene, which regulates adiponectin, plays a key role in metabolic homeostasis. Variants like rs1501299 and rs2241766 are associated with NAFLD and CVD risk. This study investigates ADIPOQ gene expression as a biomarker in early disease onset. To assess biochemical and genetic contributors to early CVD and NAFLD in young adults, with a focus on ADIPOQ gene expression and its expression to identify potential early biomarkers for prevention and intervention. In this ethically approved case-control study (Dec 2022–Dec 2024), 150 young adults with NAFLD/CVD risk and 150 matched healthy controls were recruited from clinics/camps in Thiruvananthapuram. Blood samples were analyzed at Genetika for metabolic, hormonal, oxidative, and inflammatory markers. ADIPOQ expression was assessed using qRT-PCR. Statistical tools included t-tests, ROC analysis, Firth logistic regression, and bootstrapping via Stata 17.0. Cases showed elevated fasting glucose, LDL, IL-6, and MDA, with significantly lower ADIPOQ levels. ADIPOQ expression correlated negatively with risk markers and positively with HDL and T4. MDA, urea, and low ADIPOQ emerged as strong predictors in regression analysis. Reduced ADIPOQ gene expression is linked to early CVD and NAFLD through obesity, inflammation, and oxidative stress. ADIPOQ, with MDA, HDL, and fasting glucose, may support early diagnosis. Lifestyle interventions can enhance ADIPOQ expression and reduce disease risk.

Keywords: ADIPOQ Gene Expression, Cardiovascular disease (CVD), Non-alcoholic fatty liver disease (NAFLD), Oxidative stress, Inflammation

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1. INTRODUCTION

There is a growing awareness that CVDs and NAFLD are now big health problems affecting many younger people worldwide. Previously termed NAFLD, MASLD is seen as the liver version of metabolic syndrome and may lead to or be present with heart complications [1]. Increasing rates of obesity, insulin resistance, a sedentary lifestyle, and unusual lipid levels mainly create this double stress, both for the liver and for the blood vessels [2]. Because NAFLD and CVD are becoming more common in young adults, studying the connections between their biochemistry, such as insulin resistance, chronic inflammation, and different forms of fat in the blood, is essential. Oxidative stress and slight inflammation in the body help to advance both atherosclerosis and CHD. These cytokines worsen liver inflammation and help cause fibrosis and cirrhosis in NAFLD. They also lead to problems with the lining of the blood vessels and a higher risk of atherosclerosis in CVD. Due to this, oxidative stress caused by low antioxidants compared to high reactive oxygen species worsens liver and blood vessel damage and leads to faster disease progression [3].

Adiponectin is getting a lot of focus among the various genes linked to metabolic and heart health. This protective adipokine, encrypted by the ADIPOQ gene situated on chromosome 3q27, is mainly released by adipose tissue and plays a major part in sustaining metabolic homeostasis. Adiponectin needs to regulate the way glucose and lipids are processed, to improve how insulin functions, to lower inflammation, and to reduce the risk of atherosclerosis, supporting all of this in both the liver and the heart [4]. Polymorphisms in the ADIPOQ gene, such as rs1501299 and rs2241766, notably influence its expression and serum levels. These single nucleotide polymorphisms (SNPs) are associated with alterations in lipid profiles, insulin signaling, and inflammatory responses, all of which are key contributors to the pathogenesis of NAFLD and cardiovascular diseases (CVDs) [5]. Understanding these genetic variations provides crucial insights into individual susceptibility and offers potential avenues for personalized prevention and therapeutic strategies.

In young adults, the interaction between genetic factors such as ADIPOQ gene expression and lifestyle influences, including poor diet and sedentary behavior, can trigger the early development of NAFLD and CVD. Identifying problems early, making positive lifestyle changes, and using screening can stop a disease from advancing. The study aims to understand the causes of CVD and NAFLD in young adults by studying ADIPOQ gene expression. The research aims to find a relationship between excess heart-liver problems so it can help predict risks early and offer targeted care.

1.1. NOVELTY OF THE STUDY

- **Focus on Young Adults:**
  Targets an underexplored age group to understand early-onset NAFLD and CVD, addressing the growing burden of cardiometabolic diseases in younger populations.
- **Dual Disease Model:**
  Simultaneously investigates cardiovascular events and non-alcoholic fatty liver disease, highlighting their shared biochemical and genetic pathways.
- **Emphasis on ADIPOQ Gene Expression:**
  Examines the expression of the ADIPOQ gene, providing insight into the role of adiponectin in metabolic dysfunction.
- **Integration of Biochemical and Genetic Markers:**
  Combine gene expression data with biochemical parameters such as lipid profiles and inflammatory markers to develop a comprehensive disease risk model.
- **Early Detection and Risk Prediction:**
  It aims to identify early molecular indicators of disease, enabling timely intervention through genetic screening and lifestyle modification.
- **Personalized Preventive Approach:**
  Supports individualized strategies to prevent or delay disease onset based on genetic predisposition and modifiable risk factors.

1.2. HYPOTHESIS

Altered ADIPOQ gene expressions are significantly linked to adiponectin deficiency, contributing to adipose tissue dysfunction, thereby increasing the risk of cardiovascular events and NAFLD in young adults through metabolic and inflammatory pathways.

1.3. AIM & OBJECTIVES

1.3.1 AIM

The intention of this study is to analyze the biochemical and genetic promoters to cardiovascular events and NAFLD in young adults, with a focus on ADIPOQ gene expression.

1.3.2 OBJECTIVES

- To assess the relation between ADIPOQ gene expression and the existence of cardiovascular disease and non-alcoholic fatty liver disease (NAFLD) in individuals, with a focal point on metabolic, inflammatory, and lifestyle agents.
- To analyze the normality, distribution, and comparative distinctions between cases and controls in biochemical, anthropometric, lifestyle, and clinical guidelines.
- The intention of this study is to inspect the diagnostic potential of biochemical and genetic markers, particularly ADIPOQ gene expression, malondialdehyde (MDA), lipid profiles, thyroid hormones, and inflammatory markers, using ROC curve analysis.
- To investigate the correlation of ADIPOQ gene expression with important metabolic and inflammatory biomarkers
- To assess the impact of demographic, socioeconomic, lifestyle, and dietary factors on ADIPOQ gene expression levels.
- To perform logistic regression analysis to identify independent predictors of disease status, including biochemical, inflammatory, and hormonal variables.

2. METHODOLOGY

2.1. Study Design

This case-control study was performed between December 2022 and December 2024, supporting the principles of the Declaration of Helsinki (1964). The Institutional Ethics Committee of Genetika granted ethical approval (Ref. No: 09/2022/IECG), and written informed consent was secured from all volunteers before their enrollment.

2.2 Setting

Participants were recruited from Hridayalaya Clinic, Lords Hospital, World Heart Day Medical Camp, and through referrals from various blood collection centers in Thiruvananthapuram, Kerala, India. All laboratory and genetic analyses were conducted at Genetika, Centre for Advanced Genetic Studies.

2.3 Participants

The study included 300 individuals aged 18–45. Cases (n = 150) had NAFLD and/or cardiovascular risk conditions, while controls (n = 150) were age and sex-matched healthy individuals without chronic illnesses. To minimize selection bias, both cases and controls were recruited from the same geographic region and healthcare facilities, ensuring comparability. Measurement bias was addressed by adhering to standardized protocols for biochemical and genetic assessments, with all analyses conducted blinded to participants' case-control status.

2.4. Inclusion and Exclusion Criteria

Inclusion criteria were individuals aged 18–45 years with confirmed NAFLD and/or cardiovascular risk factors.

Exclusion criteria included those outside this age range with chronic illnesses, malignancies, long-term medication use, or prior exposure to ionizing radiation, chemotherapy, or other mutagenic agents.

2.5. Data Sources and Measurement

Structured questionnaires were used to collect demographic, lifestyle, and clinical data. Fasting Venous blood (6–8 mL) was collected under fasting conditions into plain and EDTA-coated tubes.

2.6. Biochemical and Molecular Analyses

- **Fasting Blood Glucose:** Measured using the glucose oxidase-peroxidase (GOD-POD) enzymatic method.
- **Serum Triglycerides, Total Cholesterol, HDL, and LDL Cholesterol:** Assessed using standard enzymatic colorimetric methods with commercially available diagnostic kits.
- **Urea:** Assessed using the urease method.
- **Uric Acid:** Determined by the Uricase TOPS method.
- **Enzymatic Creatinine Assay:** Performed using the Jaffe's method to ensure specificity and accuracy.
- **Malondialdehyde (MDA):** Quantified as a marker of lipid peroxidation using a competitive enzyme-related immunosorbent assay (ELISA).
- **Thyroid Hormones (T3, T4, and TSH):** Estimated by chemiluminescent immunoassay (CLIA) utilizing automated analyzers.
- **Interleukin-6 (IL-6):** Measured utilizing a high-sensitivity enzyme-related immunosorbent assay (ELISA) backing the manufacturer's instructions.

Total RNA was extracted using a commercial kit and quantified by biospectrometer. cDNA was synthesized from 50 µg of RNA utilizing reverse transcriptase. Real-time PCR was performed using gene-specific primers for ADIPOQ:

Forward: 5'-CAGGCCGTGATGGCAGAGATG-3'
Reverse: 5'-GGTTTCACCGATGTCTCCCTTAG-3'

Quantitative real-time PCR (qRT-PCR) was done using ADIPOQ-specific primers on a CFX Opus 96 System (Bio-Rad, USA) with SYBR Green Master Mix (Lot No: 103024 RMM). Relative expression was measured by the 2^–ΔΔCT method using GAPDH as the internal control.

2.7. Study Size

The sample size was calculated using the formula Z^2pq/d^2, where Z is the standard normal deviation, p is the expected prevalence derived from prior studies, q is 1 – p, and d is the allowable error margin. Prevalence estimates from previously published research were used to justify the enrollment of 150 cases and 150 controls.

2.8. Statistical Methods

Descriptive statistics were computed, including means, standard deviations, and frequency distributions. Group comparisons were made using independent t-tests for normally distributed variables. ROC curve analysis assessed diagnostic performance via AUC, sensitivity, and specificity. Quantile regression examined associations between ADIPOQ expression ($2^{-\Delta\Delta CT}$) and clinical variables. Firth logistic regression was employed to evaluate the association between biomarkers and disease status, mainly due to its robustness in handling small sample sizes and rare events, which can lead to biased estimates in standard logistic regression. Levene's test was applied to assess the equality of variances between groups before conducting parametric comparisons, ensuring the assumption of homoscedasticity was met. In instances where traditional assumptions may be violated, or sample distributions appeared non-normal, bootstrap methods were used to compute more reliable p-values, enhancing the robustness of the inference drawn from the data. All analyses were conducted using Stata 17.0.

3. RESULTS

This study investigated the biochemical, endocrinological, and genetic factors contributing to cardiovascular disease (CVD) and non-alcoholic fatty liver disease (NAFLD) in young adults (aged 18–40), with a special focus on the expression of the gene ADIPOQ. Three hundred participants were enlisted, involving 150 clinically diagnosed cases (CVD or NAFLD) and 150 age- and sex-matched healthy controls.

3.1 Normality Assessment

The Shapiro-Wilk test was utilized to analyze the normality of biochemical, anthropometric, and genetic variations in both case and control groups. A p-value <0.05 indicated a significant deviation from a normal distribution. Several variables showed non-normal distributions, including duration of marriage, age at marriage, abdominal circumference, fasting blood sugar, serum triglycerides, and HDL cholesterol in cases; and serum LDL cholesterol, uric acid, enzymatic creatinine assay, malondialdehyde, T3, TSH, IL-6 in controls, and ADIPOQ gene expression in both groups.

3.2 Demographic, Socioeconomic, and Anthropometric Characteristics

The comparison of demographic and anthropometric features between cases and controls showed that sex distribution was similar ($p = 0.296$), as were birth order ($p = 0.849$), residence ($p = 0.904$), religion ($p = 0.906$), and socioeconomic status ($p = 0.386$). The educational level showed a non-significant trend toward lower attainment among cases ($p = 0.065$), while occupational type differed significantly, with sedentary occupations being more common among cases ($p = 0.007$).

Obesity was notably larger among cases (37.3%) against controls (13.3%) ($p < 0.001$), indicating a strong association with disease status. Among continuous variables, age and duration of marriage did not differ significantly, whereas age at menarche was considerably higher in cases ($p = 0.002$). Height and abdominal circumference

differences were not significant. Still, weight and BMI were notably elevated in cases ($p < 0.001$), emphasizing their role as significant risk factors for cardiovascular disease and non-alcoholic fatty liver.

3.3 Medical History and Health Conditions

The prevalence of several medical conditions differed significantly between cases and controls. Hypertension (26.7% vs. 10%), dyslipidemia (28% vs. 11.3%), coronary artery disease (14.7% vs. 0%), chest pain (21.3% vs. 0%), ischemic heart disease (14.7% vs. 0%), heart failure (11.3% vs. 0%), and cirrhosis (6.7% vs. 0%) were significantly more frequent in cases (all $p < 0.01$, except cirrhosis $p = 0.002$). Thyroid disorders (14% vs. 8.7%, $p = 0.145$) and stroke (3.3% vs. 0%, $p = 0.060$) showed no marked differences. Psychological and occupational stress was greater in cases (17.3% vs. 8%, $p = 0.015$), along with neurological symptoms: severe headaches (6.7% vs. 2%, $p = 0.047$), sudden blindness (4% vs. 0%, $p = 0.030$), and dizziness (7.3% vs. 2%, $p = 0.029$). History of COVID-19 infection was also more frequent among cases (45.3% vs. 27.3%, $p = 0.001$). Additionally, parental consanguinity was reported more often in cases (9.3% vs. 2.7%, $p = 0.015$), indicating a probable genetic tendency.

3.4 Lifestyle and Dietary Habits

Lifestyle and dietary habits differed considerably between cases and controls. Alcohol consumption was notably greater in test subjects, while regular exercise was less common. Cases showed higher rates of poor physical activity. Non-vegetarian diets and sugar restrictions were more prevalent among cases, and coconut oil was the preferred cooking oil. Cases had lower vegetable intake and differing patterns of marine fish consumption, with more reporting low or high intake than controls. Significantly, fewer cases regularly consumed home-cooked food. Smoking and tobacco chewing did not differ significantly.

3.5 Biochemical and Inflammatory Profile Differences Between Cases and Controls

Cases exhibited significantly higher fasting blood sugar, triglycerides, total cholesterol, LDL cholesterol, serum urea, uric acid, CRP, MDA, IL-6, and TSH levels compared to controls, along with lower HDL cholesterol, T3, T4, and ADIPOQ gene expression. These findings indicate pronounced metabolic imbalance, systemic inflammation, oxidative stress, and thyroid dysfunction in affected individuals.

3.6 Comparative Analysis of Clinical and Biochemical Parameters

Bootstrap analysis showed significantly higher fasting blood sugar, triglycerides, total cholesterol, LDL, urea, uric acid, creatinine, MDA, IL-6, and TSH in cases ($p < 0.01$), while HDL, T3, T4, and ADIPOQ gene expression were significantly lower. These findings suggest altered glucose and lipid metabolism, elevated inflammation and oxidative stress, thyroid dysfunction, and reduced metabolic protection in test subjects. Variance equality was assessed using Levene's test to ensure appropriate statistical comparisons.

3.7 ROC curves for various biochemical and genetic markers

Among the top-performing markers, malondialdehyde (MDA) exhibited the highest discriminative capacity with an AUC of 0.9605, followed closely by serum LDL cholesterol (AUC = 0.9262), HDL cholesterol (AUC = 0.9259), total cholesterol (AUC = 0.9063), and fasting blood sugar (AUC = 0.9046). These markers demonstrated excellent sensitivity and specificity, reinforcing their potential as reliable diagnostic indicators. In the intermediate range, interleukin-6 (AUC = 0.8589), thyroid stimulating hormone (AUC = 0.8732), tetra-iodothyronine (T4) (AUC = 0.8684), (ADIPOQ) expression (AUC = 0.8654), and tri-iodothyronine (T3) (AUC = 0.7806) showed good to strong diagnostic utility. These biomarkers maintained a well-balanced trade-off between true positive and true negative classifications and can serve as complementary tools in diagnosis. Markers such as serum triglycerides (AUC = 0.8087) and urea (AUC = 0.8087) also provided fair predictive accuracy, though their performance was slightly limited by lower specificity in some cases. Serum uric acid

displayed moderate discriminative power (AUC = 0.6913), and while it may not serve as a primary marker, it could be informative when used alongside others. Conversely, the enzymatic creatinine assay, with an AUC close to 0.5 (AUC = 0.515), showed minimal discriminatory power and was statistically non-significant, indicating that it performs no better than random classification.

3.8 Association of ADIPOQ Gene Expression with Demographic, Socioeconomic, and Obesity Variables

This analysis evaluated ADIPOQ gene expression across various demographic and socioeconomic variables in both cases and controls. Most factors, including sex ($p = 0.144$), birth order ($p = 0.745$), residence ($p = 0.678$), education ($p = 0.683$), socioeconomic status ($p = 0.660$), and religion ($p = 0.861$) did not show statistically significant associations with ADIPOQ levels. However, occupational type showed a significant association ($p = 0.006$), with non-sedentary cases displaying higher ADIPOQ expression than those in sedentary roles. Obesity was strongly associated with reduced ADIPOQ expression ($p < 0.001$), particularly in test subjects, highlighting its role in metabolic dysfunction.

3.9 Association of ADIPOQ Gene Expression with Clinical and Health Variables

ADIPOQ is linked with several medical and personal factors, as was shown by gene expression. Hypertension, dyslipidemia, thyroid problems, urinary tract infections, psychological and occupational stress, sudden severe headaches, dizziness, and a history of COVID-19 are all linked to lower levels of ADIPOQ—and this connection was observed to be significant ($p < 0.05$). The findings point to a close relationship between reduced ADIPOQ blood levels and problems with metabolism or inflammation. Yet, the results from user-reported parental consanguinity failed to link it with ADIPOQ expression. The data emphasize ADIPOQ's involvement in a person's overall health and its impact on chronic disease risk.

3.10 Association of ADIPOQ Gene Expression with Lifestyle and Dietary Habits

Lifestyle and diet strongly affect the expression of the ADIPOQ gene. People with lower levels tend to use alcohol, get less exercise, eat little fruit or vegetables, ignore their water intake, eat fewer meals at home, and choose vegetarian diets. Alcohol use, physical activity, the type of diet, and eating regular homemade meals are all strongly associated. Results showed that smoking, chewing tobacco, salt consumption, and oil have little effect. According to these results, factors in these people's lives can affect ADIPOQ expression.

3.11 Correlation of ADIPOQ Gene Expression with Metabolic and Inflammatory Biomarkers in Cases and Controls

The scatter plot analysis displays that ADIPOQ gene expression is significantly associated with various clinical and biochemical markers in both cases and controls. A consistent negative correlation was observed between ADIPOQ expression and levels of fasting blood sugar, triglycerides, total cholesterol, LDL cholesterol, urea, uric acid, creatinine, malondialdehyde (MDA), thyroid-stimulating hormone (TSH), and interleukin-6 (IL-6), with the associations being more pronounced among cases. This suggests that individuals with cardiovascular disease and NAFLD show more significant ADIPOQ downregulation alongside metabolic and inflammatory disturbances. Conversely, positive correlations were found between ADIPOQ expression and high-density lipoprotein (HDL) cholesterol and tetra-iodothyronine (T4) levels, especially among controls, suggesting better metabolic regulation in healthy individuals. These patterns highlight the potential role of adiponectin in modulating metabolic and inflammatory responses, with its reduced expression reflecting the severity of metabolic dysfunction in disease conditions.

3.12 Logistic Regression Analysis of Biochemical Markers and Case Status

In the unadjusted model, low ADIPOQ expression strongly increases case odds, but this weakens after adjustment. Fasting blood sugar remains a significant positive predictor in both models. Serum triglycerides and LDL lose significance after adjustment, while HDL consistently shows a protective effect. Serum urea and

malondialdehyde (MDA) are strong independent positive predictors, with MDA showing the highest odds. T4 is protective, TSH shows a positive association, and T3's effect is non-significant after adjustment. IL-6 shows an unadjusted positive association but reverses to a significant negative one after adjustment, indicating complex interactions.

4. DISCUSSION

This study examined biochemical, endocrine, lifestyle, and genetic factors in young adults with non-alcoholic fatty liver disease (NAFLD) and/or cardiovascular disease (CVD), focusing on ADIPOQ gene expression. Results showed significant metabolic, inflammatory, and genetic differences between cases and controls, highlighting the multifactorial nature of these diseases and the potential of ADIPOQ as a metabolic health biomarker.

4.1. Metabolic Dysregulation and Disease Risk

The elevated levels of fasting blood sugar, triglycerides, total cholesterol, LDL cholesterol, serum urea, uric acid, malondialdehyde (MDA), interleukin-6 (IL-6), and thyroid-stimulating hormone (TSH) in cases indicate profound metabolic derangement, systemic inflammation, oxidative stress, and thyroid dysfunction. These findings align with previous studies linking metabolic syndrome components and systemic inflammation to increased CVD and NAFLD risk [6, 7]. Conversely, protective markers such as HDL cholesterol, triiodothyronine (T3), thyroxine (T4), and ADIPOQ were markedly lower in cases, reinforcing their role in maintaining metabolic equilibrium.

4.2. ADIPOQ Gene Expression and Disease Pathophysiology

ADIPOQ expression was significantly reduced in cases, consistent with prior studies reporting adiponectin as an anti-inflammatory, insulin-sensitizing adipokine whose deficiency is implicated in obesity, atherosclerosis, and hepatic steatosis [8]. The robust negative correlation of ADIPOQ with fasting blood sugar, triglycerides, LDL cholesterol, MDA, IL-6, and TSH highlights its inverse relationship with metabolic stress and inflammation. Its positive association with HDL cholesterol and T4 further underscores its role in lipid and endocrine homeostasis. Logistic regression identified low ADIPOQ levels as a strong unadjusted predictor of disease status. Although this association weakened upon adjustment for other variables, the persistence of altered

ADIPOQ expression in conjunction with elevated MDA and urea underscores its potential as part of a multi-marker panel for early identification of at-risk individuals.

4.3. Lifestyle, Diet, and ADIPOQ Expression

Lifestyle factors such as physical inactivity, alcohol use, low intake of fruits and vegetables, and infrequent consumption of home-cooked meals were all significantly associated with reduced ADIPOQ expression. These findings agree with previous studies linking poor dietary habits and sedentary behavior with lower ADIPOQ levels and greater cardiometabolic risk [9]. Interestingly, a vegetarian diet was also associated with lower ADIPOQ expression in this cohort, possibly reflecting imbalanced or protein-deficient dietary patterns rather than vegetarianism.

4.4. Inflammation, Oxidative Stress, and ADIPOQ

Elevated MDA and IL-6 levels in cases support the presence of oxidative and inflammatory stress, hallmarks of both CVD and NAFLD. In ROC analysis, the high discriminative power of MDA (AUC = 0.9605) and IL-6 (AUC = 0.8589) further underscores their diagnostic value. ADIPOQ's negative correlation with these markers suggests a compensatory downregulation or impaired secretion under chronic metabolic stress.

4.5. Endocrine Dysfunction and ADIPOQ

Altered thyroid hormone levels, specifically increased TSH and decreased T3/T4 in cases, were significantly correlated with ADIPOQ expression. These findings support a functional interaction between adipokines and thyroid hormones, where hypothyroid states may downregulate ADIPOQ, contributing to further metabolic dysfunction [10].

4.6. Sociodemographic and Occupational Influences

While most sociodemographic factors were not associated with ADIPOQ expression, occupational type showed a significant association with sedentary work linked to lower expression. This aligns with evidence showing reduced physical activity impairs adiponectin secretion and worsens insulin sensitivity [11].

4.7. Clinical Implications and Biomarker Potential

The ROC analysis demonstrated that several markers, including MDA, LDL cholesterol, HDL cholesterol, total cholesterol, and fasting glucose, have excellent discriminative power, supporting their use in early screening. ADIPOQ, with an AUC of 0.8654, showed strong diagnostic potential and could be considered a complementary tool alongside traditional markers in risk stratification protocols.

4.8. Strengths and Limitations

The study's strengths include a well-matched case-control design, comprehensive profiling of clinical, biochemical, lifestyle, and genetic variables, and the use of robust statistical techniques, including ROC and logistic regression analysis. However, it has certain limitations. Further longitudinal studies are needed to determine ADIPOQ's predictive capacity and utility in monitoring disease progression or response to intervention.

5. SUMMARY AND CONCLUSION

5.1. Summary

- **ADIPOQ gene expression** was significantly lower in cases and negatively correlated with metabolic and inflammatory markers.

- **Obesity**, sedentary lifestyle, and poor dietary practices were significantly more common among cases and linked to lower ADIPOQ expression.

- **Biochemical markers** like fasting blood sugar, triglycerides, LDL, urea, uric acid, MDA, and TSH were significantly elevated, while HDL, T3, T4, and ADIPOQ were lower in cases.

- **ROC analysis** showed that MDA, LDL, HDL, and fasting blood sugar are excellent discriminators between cases and controls.

- **Logistic regression** identified MDA and serum urea as strong independent predictors of disease status, while HDL and T4 showed a protective effect.

- **ADIPOQ expression** is a promising candidate biomarker for early identification of metabolic dysregulation.

5.2. Conclusion

This study highlights the intricate relationships between genetic expression, biochemical profiles, and lifestyle factors in young adults having cardiovascular disease and non-alcoholic fatty liver disease. The decline in ADIPOQ gene expression is strongly linked with obesity, metabolic abnormalities, inflammation, and oxidative stress. Integrating ADIPOQ levels with biomarkers such as MDA, HDL, fasting blood sugar, and thyroid hormones could be a valuable diagnostic tool in early risk

stratification and disease management. Notably, the strong influence of modifiable lifestyle and dietary habits on ADIPOQ expression underscores the potential for preventative strategies focused on nutrition, physical activity, and weight management to mitigate disease risk in vulnerable populations.

6. REFERENCES

1. Eslam, M., Newsome, P. N., Sarin, S. K., Anstee, Q. M., Targher, G., Romero-Gomez, M., Zelber-Sagi, S., Wai-Sun Wong, V., Dufour, J. F., Schattenberg, J. M., Kawaguchi, T., Arrese, M., Valenti, L., Shiha, G., Tiribelli, C., Yki-Järvinen, H., Fan, J. G., Grønbæk, H., Yilmaz, Y., Cortez-Pinto, H., … George, J. (2020). A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. Journal of hepatology, 73(1), 202–209. https://doi.org/10.1016/j.jhep.2020.03.039
2. Matyas, C., Haskó, G., Liaudet, L., Trojnar, E., & Pacher, P. (2021). Interplay of cardiovascular mediators, oxidative stress and inflammation in liver disease and its complications. Nature reviews. Cardiology, 18(2), 117–135. https://doi.org/10.1038/s41569-020-0433-5
3. Moghtadaie, A., Mahboobi, H., Fatemizadeh, S., & Kamal, M. A. (2023). Emerging role of nanotechnology in treatment of non-alcoholic fatty liver disease (NAFLD). EXCLI journal, 22, 946–974. https://doi.org/10.17179/excli2023-6420
4. Francischetti, E. A., Dezonne, R. S., Pereira, C. M., de Moraes Martins, C. J., Celoria, B. M. J., de Oliveira, P. A. C., & de Abreu, V. G. (2020). Insights into the controversial aspects of adiponectin in cardiometabolic disorders. *Hormone and Metabolic Research, 52*(10), 695–707. https://doi.org/10.1055/a-1239-4349
5. Saki, S., Saki, N., Poustchi, H., & Malekzadeh, R. (2020). Assessment of Genetic Aspects of Non-alcoholic Fatty Liver and Premature Cardiovascular Events. Middle East journal of digestive diseases, 12(2), 65–88. https://doi.org/10.34172/mejdd.2020.166
6. Al Rifai, M., Silverman, M. G., Nasir, K., Budoff, M. J., Blankstein, R., Szklo, M., Katz, R., Blumenthal, R. S., & Blaha, M. J. (2015). The association of nonalcoholic fatty liver disease, obesity, and metabolic syndrome, with systemic inflammation and subclinical atherosclerosis: the Multi-Ethnic Study of Atherosclerosis (MESA). Atherosclerosis, 239(2),629–633. https://doi.org/10.1016/j.atherosclerosis.2015.02.011
7. Schwimmer, J. B., Pardee, P. E., Lavine, J. E., Blumkin, A. K., & Cook, S. (2008). Cardiovascular risk factors and the metabolic syndrome in pediatric nonalcoholic fatty liver disease. Circulation, 118(3), 277–283. https://doi.org/10.1161/CIRCULATIONAHA.107.739920
8. Choi, H. M., Doss, H. M., & Kim, K. S. (2020). Multifaceted Physiological Roles of Adiponectin in Inflammation and Diseases. International journal of molecular sciences, 21(4), 1219. https://doi.org/10.3390/ijms21041219
9. Agostinis-Sobrinho, C., Moreira, C., Abreu, S., Lopes, L., Oliveira-Santos, J., Steene-Johannessen, J., Mota, J., & Santos, R. (2017). Serum Adiponectin Levels and Cardiorespiratory Fitness in Nonoverweight and Overweight Portuguese Adolescents: The LabMed Physical Activity Study. Pediatric exercise science, 29(2), 237–244. https://doi.org/10.1123/pes.2016-0162

10. Delitala, A. P., Scuteri, A., Fiorillo, E., Lakatta, E. G., Schlessinger, D., & Cucca, F. (2019). Role of Adipokines in the Association between Thyroid Hormone and Components of the Metabolic Syndrome. Journal of clinical medicine, 8(6), 764. https://doi.org/10.3390/jcm8060764
11. Kriketos, A. D., Gan, S. K., Poynten, A. M., Furler, S. M., Chisholm, D. J., & Campbell, L. V. (2004). Exercise increases adiponectin levels and insulin sensitivity in humans. Diabetes care, 27(2), 629–630. https://doi.org/10.2337/diacare.27.2.629

7. ANNEXURES

7.1 LIST OF PUBLICATIONS

1. Raveendran, R., Josephine, A., Kalaiselvi, P., Sureka, V., Kanchana, S. B., Abdulla, T., & Others. (2023). Multifactorial aspects influencing non-alcoholic fatty liver disease (NAFLD). Journal of Advanced Zoology, 44(S-1), 1059–1072. https://doi.org/10.17762/jaz.v44iS-1.1248
2. Rachana Raveendran, Anthony J, Sureka, V, Kalaiselvi, P, Varma, P. R. R, Kandy, L. N, Nair, R. K. R, Kottola, M, Mohanan, M. T, Thoduvayil, S, Abdulla, T, Kottiyath, P, & Divakaran, D. R. (2025). Association of oxidative stress, inflammation, and ADIPOQ gene expression in young adults at risk of non-alcoholic fatty liver disease and cardiovascular disease [Manuscript accepted for publication].

7.2 PROOF OF PUBLICATIONS

Journal of Advanced Zoology

ISSN: 0253-7214
Volume ***44*** *Issue* ***S-1 Year 2023*** *Page* ***1059:1072***

Multifactorial Aspects Influencing Non-Alcoholic Fatty Liver Disease (Nafld)

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Article History

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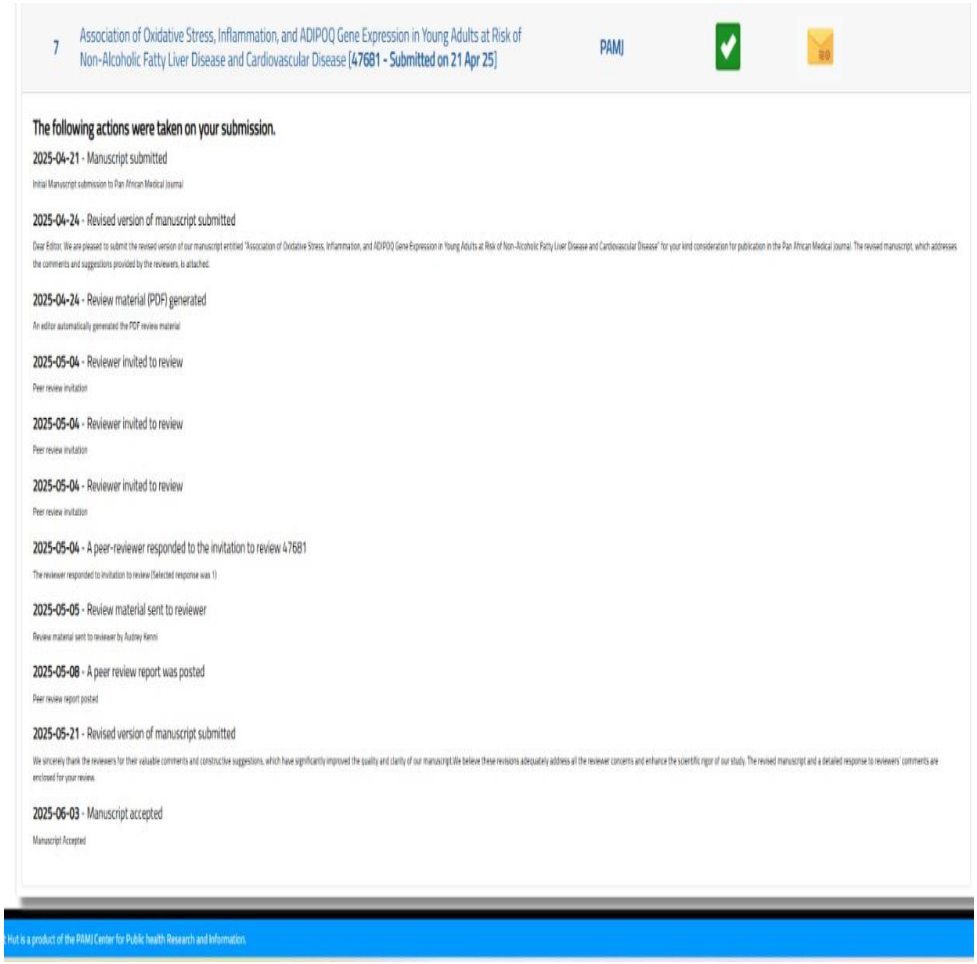
Abstract

Nonalcoholic fatty liver disease (NAFLD) is a growing public health concern, with a prevalence of up to 25% worldwide. While once considered a benign condition, NAFLD is now recognized as a major cause of chronic liver disease, liver failure, and hepatocellular carcinoma. The pathogenesis of NAFLD is multifactorial and involves a complex interplay between genetic, environmental, and metabolic factors. In this review, we provide an overview of the multifactorial aspects of NAFLD, including genetic predisposition, insulin resistance, dyslipidemia, gut microbiota, dietary factors, and physical inactivity. We also discuss the role of inflammation, oxidative stress, and hepatic steatosis in the progression of NAFLD to nonalcoholic steatohepatitis (NASH), cirrhosis, and hepatocellular carcinoma. Finally, we review the current and emerging therapies for NAFLD and NASH, including lifestyle modifications, pharmacological interventions, and surgical approaches. The multifactorial nature of NAFLD requires a comprehensive approach to diagnosis, treatment, and prevention, with a focus on addressing the underlying metabolic and environmental factors that contribute to its development and progression.

Keywords: *Non-Alcoholic Fatty Liver Disease; metabolic associated fatty liver disease; Nonalcoholic steatohepatitis; diabetics; Genetic factors; Thyroid dysfunction*

1. Introduction

Non-alcoholic fatty liver disease (NAFLD) is a term used to describe a prevalent clinicopathological illness in which patients do not have a history of drinking too much alcohol. The prevalence of NAFLD in the general population ranges from 10 to 39% [1]. According to Arab et al. [2], "NAFLD is defined as at least 5% steatosis observed in the hepatocytes on either histology or by imaging methods such as proton density fat fraction (PDFF)". Tarantino et al. [3] explained that, "NAFLD is an increasingly recognized condition, linked to metabolic syndrome (MS)". Green and Hodson [4] revealed that, "according to current theories, the NAFLD is a condition which is characterised by an excessive hepatic buildup of lipids by an enhanced input/output balance of fatty acids and because of high calorie intake, sedentarism, and MS, high energy diets cause the liver to absorb more free fatty acids, by lipolysis and de novo hepatic lipogenesis. Yet, the output mechanisms associated with fatty acid oxidation and the release of very low-density lipoproteins (VLDL) is still insufficient to prevent the accumulation of triglycerides".



7 Association of Oxidative Stress, Inflammation, and ADIPOQ Gene Expression in Young Adults at Risk of Non-Alcoholic Fatty Liver Disease and Cardiovascular Disease [47681 - Submitted on 21 Apr 25] PAMJ

The following actions were taken on your submission.

2025-04-21 - Manuscript submitted

Initial Manuscript submission to Pan African Medical Journal

2025-04-24 - Revised version of manuscript submitted

Dear Editor, We are pleased to submit the revised version of our manuscript entitled 'Association of Oxidative Stress, Inflammation, and ADIPOQ Gene Expression in Young Adults at Risk of Non-Alcoholic Fatty Liver Disease and Cardiovascular Disease' for your kind consideration for publication in the Pan African Medical Journal. The revised manuscript, which addresses the comments and suggestions provided by the reviewers, is attached.

2025-04-24 - Review material (PDF) generated

An editor automatically generated the PDF review material

2025-05-04 - Reviewer invited to review

Peer review invitation

2025-05-04 - Reviewer invited to review

Peer review invitation

2025-05-04 - Reviewer invited to review

Peer review invitation

2025-05-04 - A peer-reviewer responded to the invitation to review 47681

The reviewer responded to invitation to review (Selected response was 1)

2025-05-05 - Review material sent to reviewer

Review material sent to reviewer by Audrey Kenni

2025-05-08 - A peer review report was posted

Peer review report posted

2025-05-21 - Revised version of manuscript submitted

We sincerely thank the reviewers for their valuable comments and constructive suggestions, which have significantly improved the quality and clarity of our manuscript.We believe these revisions adequately address all the reviewer concerns and enhance the scientific rigor of our study. The revised manuscript and a detailed response to reviewers' comments are enclosed for your review.

2025-06-03 - Manuscript accepted

Manuscript Accepted

Hut is a product of the PAMJ Center for Public health Research and Information.

Association of Oxidative Stress, Inflammation, and ADIPOQ Gene Expression in Young Adults at Risk of Non-Alcoholic Fatty Liver Disease and Cardiovascular Disease

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Structured Abstract (Final Version Provided Below)- Word count: 250 words

Original Full Text - Word count: 3718

Abstract

Background:

Non-alcoholic fatty liver disease (NAFLD) and cardiovascular diseases (CVD) are increasingly seen in young adults due to obesity, sedentary lifestyle, and poor diet. Oxidative stress and chronic inflammation are key contributors to their development, promoting liver damage, endothelial dysfunction, and atherosclerosis. Adiponectin, encoded by the *ADIPOQ* gene, has protective metabolic effects, and its reduction—often influenced by genetic factors—is linked to increased disease risk. This study evaluates the relationship between oxidative stress (MDA), inflammation (IL-6), and *ADIPOQ* gene expression in young adults to identify early biomarkers.

7.3 ETHICAL CERTIFICATE

Genetika
Centre for Advanced Genetic Studies

INSTITUTIONAL ETHICS COMMITTEE OF GENETIKA

Registered and approved by NECRBHR, Department of Health Research (DHR)

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M.Sc, Ph.D

Ms. CS SUJA DEVI BA

Letter of Approval

Ref. No. 09/2022/IECG Date : 12/12/2022

The Institutional Ethics Committee of Genetika, reviewed and discussed the Ph.D Research Proposal of **Mrs. Rachana Raveendran** of Meenakshi Academy of Higher Education and Research titled "**Biochemical and Genetic aspects in the Pathogenesis of Cardiovascular events and Non-alcoholic Fatty Liver in Young adults with special emphasis on Adiponectin encoding (ADIPOQ) Gene Polymorphism**".

The following documents were reviewed:

1. Research Proposal, Protocol and Synopsis.
2. Patient Information Sheet and Informed Consent Form in English and local language (Malayalam).

We are pleased to inform you that your above Project proposal (No.09/2022/IECG) was approved in its present form by the IECG in the meeting held on 27/11/2022, subject to following conditions.

1. The IECG expects to be informed about the progress of the study in every six months.
2. You are directed to coordinate with the Physicians on sample collection in the present study as per GCP guidelines and respective hospital SOP.
3. Any Changes / Modifications / Alterations / Deviations in the study protocol have to be notified to the Ethics Committee for consideration.
4. If the study is discontinued / abandoned at any stage before its completion, the same shall be communicated to the Ethics Committee in writing with a copy of completed report of the activities.
5. A copy of the final report / publications of the study should be provided to the Ethics Committee.

Chairman Member Secretary

GENETIKA CENTRE FOR ADVANCED GENETIC STUDIES THIRUVANANTHAPURAM
INSTITUTIONAL ETHICS COMMITTEE

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PLAGIARISM VERIFICATION CERTIFICATE

Date:23/09/2025

This is to certify that the synopsis of the Ph.D. thesis titled **Biochemical and Genetic Aspects in The Pathogenesis of Cardiovascular Events and Non-Alcoholic Fatty Liver in Young Adults with Special Emphasis on Adiponectin Encoding (Adipoq) Gene Expression** submitted by **RACHANA RAVEENDRAN** (Reg. No: **21901008**), research scholar has been checked for plagiarism using Turnitin professional software. The similarity index recorded is **10%**, which is well within the acceptable limits as per institutional and academic guidelines. The Doctoral Committee has verified the report on plagiarism check with the contents of the synopsis and appropriate measures have been taken to ensure originality of the research accomplished herein.

A. Josephine 24/11/25

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DECLARATION OF ORIGINALITY OF RESEARCH WORK

Date:23/09/2025

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Session	:	**July 2021**
Faculty	:	**Medicine**
Department	:	**Biochemisrty**
Category	:	Part Time (External)
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We hereby declare that,

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4. The Doctoral Committee has verified the report on plagiarism check with the contents of the synopsis
5. The soft copy submitted for plagiarism check is the same as the print copy of the synopsis submitted to the Controller of Examinations, MAHER

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